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Formation of Pseudoaneurysm after Aortic Valve Replacement without Previous Endocarditis: A Case – Control Study

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Abstract

Objective: To identify the predisposing factors for pseudoaneurysm formation after aortic valve replacement without previous endocarditis.

Methods: Echocardiography was used to identify patients. Parameters with influence on the occurrence of pseudoaneurysms were analyzed and the odds ratio for the influence of the type of valve was estimated. The Chi-squared goodness-of-fit test was used to analyze if either location or underlying etiology are associated with an accumulated occurrence of a pseudoaneurysm. Fisher's Exact test was used to assess a possible relation between occurrence of a pseudoaneurysm after composite graft implantation and etiology or location.

Results: Patients treated with a composite graft had a 27-fold increased risk to develop a pseudoaneurysm ($\psi_{MH}=27$, 95%-CI: 1.61-454.19) in comparison to aortic valve replacement only. There was a significant difference for the probability of different etiologies to occur ($p=0.032$), with Stanford type A aortic dissection and aortic regurgitation being the most often occurring pathologies. A significant association between the use of a composite graft and both the underlying etiology ($p=0.002$) and the location of the pseudoaneurysm ($p=0.04$) was found. Furthermore, patients with a composite graft had a larger diameter of the aortic root as compared to patients with aortic valve replacement only ($p=0.03$). Neither the diameter of the annulus of the aortic valve ($p=0.41$; 95%-CI: 0.89-1.32) nor the diameter of the ascending aorta ($p=0.54$; 95%-CI: 0.27-1.97) had any influence on pseudoaneurysm formation.

Conclusions: The underlying disorder, determining the surgical procedure, influences the risk for the development of a pseudoaneurysm in patients without previous

Pseudoaneurysm after aortic valve replacement

endocarditis. Location of most pseudoaneurysms at the level of the aortic root may be a consequence of its larger diameter.

Keywords: pseudoaneurysm, aorta, aortic valve replacement, composite graft, homograft

Introduction

Pseudoaneurysm (false aneurysm, PA) formation is a potential late complication after aortic valve replacement (AVR) with potentially fatal consequences including high rates of rupture, recurrence and sepsis.¹ Predisposing factors for PA formation are dissection of the native aorta, infection, connective tissue disorders, preoperative chronic hypertension and aortic calcification.² Echocardiography, CT and MRI are the non-invasive investigations of choice to diagnose PA.³ Although PA formation is a well-known complication, no data exist about predisposing factors leading to non-infective PA development after AVR. Hence, the aim of the present study was to determine these factors in our collective of patients after AVR.

Methods

Definition of pseudoaneurysm: PA was defined as a rupture of the aortic wall or rupture of the mitral-aortic intravalvular fibrosa, with the free wall of the pseudoaneurysm being made of fibrous tissue and not the aortic wall, per se. The communication between the perfused echo-free space within the native aortic wall due to partial dehiscence at the suture line and the left ventricular outflow tract was visualized as a systolic-diastolic color Doppler signal in transthoracic echocardiography (**Figure 1A and 1B**).¹ Typically, the onset of this signal within the echo-free space occurred before the onset of the systolic color Doppler signal within the aortic vessel wall.

Patient population: We retrospectively evaluated all echocardiographic reports in adults (>16 years old) performed in our echocardiography laboratory during a 15-year-period (1992-2007) in order to identify patients with PA formation after AVR. Echocardiograms were carefully reviewed to confirm the diagnosis of PA before inclusion of the patients into the study. Criteria for the inclusion into the study were a preoperative echocardiography performed in our laboratory, implantation of a biological or mechanical aortic valve (AV) prosthesis and no evidence for endocarditis prior to or after AVR. In a case-control design, all patients with PA after AVR were matched with controls with respect to age, gender and time of operation. To this end, a specifically designed computer program had been used. The patients' history, the pre- and postoperative clinical data and reports from the operation were obtained from the medical and surgical records in all cases and controls. The presence of a PA was compared with the type and size of the prosthesis implanted, the diameter of the ascending aorta, of the aortic root and of the aortic annulus.

Color-coded Doppler echocardiography: Doppler echocardiography was performed according to standard techniques using a real-time phased array sector scanner with integrated color Doppler facilities (3.5 MHz). Preoperative echocardiography included the determination of the aortic valve function, the measurement of the annulus size of the AV, the diameter of the ascending aorta and of the aortic root. Measurements of the annulus size were performed in the two-dimensional long axis view of the left ventricle and of the left ventricular outflow tract. Measurements of the ascending aorta and of the aortic root were performed in the two-dimensional long axis view of the aortic root and of the ascending aorta, respectively, using M-Mode echocardiography. The postoperative development of a PA was defined as a perfused, echo-free space communicating with the left ventricular outflow tract as described above.

Operative Technique for AVR: The heart was exposed through a median sternotomy. After heparinisation the ascending aorta and right atrium were cannulated, and cardiopulmonary bypass (CPB) was initiated. The patients were cooled to 30°C. The aorta was crossclamped, and blood cardioplegia was infused antegradely and retrogradely. The aortic valve was exposed through an oblique/transverse aortotomy. The native aortic valve was excised and the annulus thoroughly decalcified. The selection of the correct size of the aortic valve prosthesis was performed on the basis of the preoperatively measured annulus size and by sizing of the annulus using appropriate valve sizers. Valve implantation was typically in a supra-annular position, using a non-everting suture technique with 2.0 Ticron suture (Ethicon, Johnson&Johnson Inc, New Jersey, USA). Rewarming was started, aortotomy was closed and the aorta declamped. The patients were weaned from CPB, and the cannulae were removed. Protamine was administered, and the operation was

terminated in a standardized fashion. The operative technique described here did not substantially change over the observation period of our study.

Statistical analyses: The analyses were done using the statistical software package R version 2.7.2. for Mac OS X. A case-control study design with 1:1 matching for age and gender was selected. The influence of the type of valve on the development of PA was examined after grouping the implanted aortic valves into the following categories: Mechanical prostheses, biological prostheses, homografts, and composite grafts. The Mantel-Haenszel estimator was used to estimate the odds ratio for the influence of the type of valve. The diameters of the aortic root and of the ascending aorta were compared between AVR and composite grafts using the nonparametric Mann-Whitney-test. Furthermore, differences of the annulus size, as determined preoperatively by echocardiography, and of the size of the implanted AV prosthesis were compared between patients who developed PA and controls using conditional logistic regression analysis. To analyze differences between the different locations and etiologies leading to pseudoaneurysms, the Chi Squared Goodness-of-fit test for equal probabilities was used. To assess if there is a relation between the occurrence of PA after composite graft implantation and etiology or location of PA, Fisher's Exact Test was used. A p-value of < 0.05 was regarded as statistically significant, and 95%-confidence intervals were calculated.

Results

Patient population

During a 15-year-period from 1992 until 2007, 24 patients fulfilling the inclusion criteria were identified and matched to controls. PA was detected in 13% (n=3) of the patients after implantation of a homograft, in 29% (n=7) of the patients after implantation of a mechanical or biological valve prosthesis and in 54% (n=14) of the patients after implantation of a composite graft. Mean age at diagnosis was 52 years (range: 20-87 years). Eighteen patients were male, six female (**Table 1**). 38% of the patients (n=9) had undergone previous aortic valve surgery. There was no significant difference in the concomitant occurrence of arterial hypertension between cases (38%, n=9) and controls (25%, n=6) (p=0.16; ns). There were two patients in the case group with Marfan syndrome, whereas in the control group one patient with Marfan syndrome and one patient with Shone's complex could be identified. The mean time from operation to first documentation of PA was 705 ± 1482 days (range 1-6289 days, median 68 days).

Influence of a mismatch between AV annulus diameter and aortic prosthesis size

To analyze the influence of the relation between AV annulus diameter preoperatively assessed by echocardiography and the size of the implanted aortic valve, the differences between the AV annulus diameter and the aortic valve size were calculated. The median difference was 1 mm (range: -5 mm to +8 mm) in the PA group and 0 mm (range: -6 mm to +5 mm) in the control group. In five patients with pseudoaneurysm and in four patients of the control group, there was no difference between the AV annulus and the implanted aortic valve size at all. Overall, no significant difference was detected between AV annulus diameter and the size of the implanted aortic valve (p=0.74; 95%-CI: 0.21-2.98 for odds ratio of 0.8). In 24% of the

patients in the case group (n=6) and in 67% of the patients in the control group (n=16) calcifications of the aortic annulus were detected.

Influence of the type of valve

The type of prosthesis (i.e. mechanical or biological prosthesis) had no significant influence on the development of PA ($p=0.74$; 95%-CI: 0.34-4.65). We also tested the influence of the type of valve implanted (AVR (n=7) or composite graft (n=14)). The Mantel-Haenszel estimator revealed that patients treated with a composite graft had a steeply (27-fold) increased risk to develop PA after AVR ($\psi_{MH}=27$; 95% CI: 1.61-454.19). Due to the low number of patients that received a homograft (n=3), these patients had been excluded from the analysis.

Influence of preexisting pathologies

The indication for valve replacement was based on the following etiologies: aortic valve insufficiency in 8 patients, aortic dissection (Stanford classification A) in 7 patients, combined stenotic and insufficient tricuspid aortic valve in 4 patients and a combined stenotic and insufficient bicuspid aortic valve in 2 patients. Rupture of the sinus of valsalva and aneurysm of the ascending aorta was documented in one patient each. The Chi Squared Goodness-of-fit test revealed a significant difference between the probabilities of different etiologies ($p=0.032$).

Influence of the location of the pseudoaneurysms

In 9 patients the PA was located at the non-coronary aortic sinus, in 6 patients the PA was located at the right coronary aortic sinus and in 3 patients at the left coronary aortic sinus. In 2 patients the PA was located at the ascending aorta and in 4 patients, a combination of the locations was detected. However, we did not find any

difference between the probability of the occurrence of a PA at these locations ($p=0.170$).

Influence of location and etiology on the occurrence of pseudoaneurysms after composite graft implantation

A composite graft was implanted in 14 patients. In 7 patients the composite graft was implanted after aortic dissection (Stanford classification A), in 4 patients a composite graft was implanted for aortic insufficiency. Rupture of the sinus of Valsalva and aneurysm of the ascending aorta was the underlying pathology and the indication for replacement with a composite graft in one patient each. We detected a significant association between the use of a composite graft and the underlying etiology ($p=0.002$). Furthermore, there was also a significant association between the location of PA and the implantation of a composite graft ($p=0.04$). PA located at the non-coronary and at the right coronary aortic sinus occurred more often than at other sites.

Influence of the diameter of the AV annulus, the aortic root and the ascending aorta

Mean diameter of the aortic root, obtained before surgery, was 56 ± 16 mm (range 26-99 mm) in patients with composite grafts, which was significantly larger ($p=0.03$) than the corresponding mean diameter in patients with AVR (38 ± 6 mm; range 31-39 mm) (**Figure 2A**). Mean diameter of the ascending aorta was 39 ± 8 mm (range 29-61 mm), and did not statistically significant differ from that of patients with AVR (36 ± 5 mm; range 28-42 mm) ($p=0.852$; ns) (**Figure 2B**).

We then tested the influence of the diameter of the annulus of the ascending aorta and of the aortic root on the development of PA. However, neither the diameter of the AV annulus ($p=0.41$; 95%-CI: 0.89-1.32) nor the diameter of the ascending aorta

($p=0.54$; 95%-CI: 0.27-1.97) had any influence on PA formation in the logistic regression analysis. However, there was a trend towards an influence of the diameter of the aortic root on PA formation ($p=0.051$; 95%-CI: 0.99-5.63). With a p-value only slightly above the significance level, there is certainly a tendency towards the development of PA in the presence of larger diameters of the aortic root.

Discussion

We found in this case-control study a close association between implantation of a composite graft and the development of a PA in patients without previous endocarditis. Furthermore, among all patients with PA, those with a composite graft had a significantly larger aortic root than those who received an AVR only. When eventually seeking to determine possible variables which might have an influence on the occurrence of a PA in this setting, and which may therefore shed light on the underlying mechanisms, we again found a higher diameter of the aortic root, but not of the ascending aorta nor of the aortic valve annulus, as a possible determinant. However, although there was a strong tendency for a PA to develop in the presence of larger diameters of the aortic root, this trend failed to reach statistic significance. Most interestingly, we also found a significant difference for the probability of the different etiologies to occur, with Stanford type A aortic dissection and aortic regurgitation (irrespective of its underlying reason) being the most often occurring pathologies. Furthermore, a significant association between the implantation of a composite graft and both the underlying etiology and the location of the PA could also be detected. PA occurred most often at the non-coronary and at the right coronary aortic sinus. This means that the underlying disorder determines the eventually performed surgical procedure, which, in turn, influences the risk that a PA will develop. Obviously, one has to admit that insertion of a composite graft carries an inherent risk of PA formation, as there are more suture lines (annulus, coronary artery anastomoses, or distally in the aortic arch) as compared with AVR only. However, analysis of the location of PA revealed that all but two PAs, which were more distal in the ascending aorta, were at the level of the aortic root. Therefore, also taking into account that an extremely experienced echocardiographer (R.J.) looked thoroughly for PA at all sites at which they may have occurred, we hypothesize that

the fact of an increased diameter of the aortic root in these patients may have a distinct pathophysiological meaning, possibly via an increased likelihood of intimal rupture.

Several morphological changes of the aortic annulus can be found after AVR. Hence, most of these findings are incidental and innocuous in nature. PA formation occurs rather rarely after AVR, but it is important to recognize this complication after aortic repair to improve late outcome.⁴ Although the clinical symptoms associated with PA formation may be non-specific or even completely absent, some patients may be severely limited by dyspnoea and fatigue, probably secondary to reduced cardiac output and/or left ventricular volume overload.⁵ Furthermore, as there is the imminent possibility of aortic rupture, early diagnosis of aortic pseudoaneurysm is essential.⁵

The preoperative assessment of the diameter of the aortic valve annulus allows improved matching of patients and valve prosthesis and, thereby, yields improved long-term results after AVR.⁶ Hence, preoperative transthoracic and transoesophageal echocardiography accurately predict the size of aortic valve prosthesis and therefore may reduce CPB time.⁷ However, our data indicate that a difference between the annulus size initially measured in preoperative echocardiography and the size of the aortic valve eventually implanted had no influence on PA formation. Somewhat surprisingly, our patients with PA formation had aortic annuli that were less calcified than those of control patients. Therefore, our data suggest that calcifications of the aortic annulus have no effect on PA formation.

The development of PA is a complication following composite graft replacement,^{5, 8} and homograft replacement¹ of the aortic valve and the ascending aorta. Kouchoukos found nine patients with PA in a study of 127 patients with composite grafts,⁸ Barbetseas described eight patients with PA of the ascending aorta in 35

patients with composite aortic grafts,⁵ and Oechslin found a PA in 22 out of 30 patients after homograft implantation.¹

In the study showing that patients after homograft implantation have a greater risk to develop PA,¹ patients with previous endocarditis were included, while these patients were excluded in our study. Indeed, PAs were often localized at the site of an abscess or a paravalvular leak after prosthetic valve endocarditis. In contrast to this study, we specifically aimed at identifying underlying factors for the development of PA in the absence of endocarditis.

Barbetseas and colleagues previously found eight patients with PA of the ascending aorta after composite graft replacement in a series of 35 patients with composite aortic grafts.⁵ Unlike our patients, most of these patients (seven out of eight) had a major risk factor (four with Marfan, two with hypertension, one with Marfan and hypertension). They found an increased diameter (mean 90 ± 30 mm, range 60-140 mm) of the ascending aorta in patients who developed PA as compared with those without PA (mean 42 ± 6 mm, range 32-50 mm). In contrast, in our study, the diameter of the ascending aorta was much smaller (mean 38 ± 8 mm, range 25-61 mm) and did not significantly differ from the diameter in the control group (mean 38 ± 5 mm, range 27-48 mm). However, we found in our patients with PA the aortic root enlarged as compared with controls (mean 48 ± 19 mm, range 26-99 mm vs. mean 37 ± 6 mm, range 28-54 mm), and there was a striking trend towards an increased risk for the development of PA in patients with an enlarged aortic root. This also holds true when only our two patients with PA localized to the ascending aorta are taken into account (aortic root: 51 and 74 mm; ascending aorta: 47 and 30 mm).

While postoperative morphological changes of the aortic annulus can be found after AVR, most of them are innocuous. The development of a pseudoaneurysm is,

however, possible and is certainly an underrecognised complication, which may lead to serious consequences.

Most importantly, our data also suggest that baseline pathology predisposes to the development of postoperative PA. This may imply that the underlying disease (i.e. Stanford type A dissection and aortic regurgitation, irrespective of its etiology), necessitating composite grafting, and not only just the type of operation (i.e. implantation of a composite graft) leads to an increased incidence of PA.

Although probably underrecognized, especially noninfectious PAs seem to be a rather rare complication. This is underlined by the fact that nearly 3700 aortic valve operations were performed at our institution during the observation period. The incidence of noninfectious PA was below 1%. However, it increased to around 4% in composite grafts and homografts, respectively. From our experience, unlike the management of PA in the setting of a postoperative infection, watchful waiting with close echocardiographic controls is the appropriate procedure for noninfectious PA. Normally, transthoracic echocardiography should be sufficient, at least in patients with good image quality. In the series presented here, no patient had to undergo re-operation for PA during a follow-up period of several years. As most PAs only develop a long time after operation (see results), postoperative follow-up of all patients, irrespective of the type of operation, may initially be the same.

Taken together, in the setting of AVR without previous endocarditis, patients with a composite graft replacement have an increased risk to develop PA, while patients having undergone AVR only have not. Patients with composite grafts developing PA are characterized by an increased aortic root. As patients with an increased diameter of the aortic root tended to have an increased risk to develop PA in general, one may assume that the type of valve together with the diameter of the aortic root are among the key parameters to be taken into account in the preoperative risk assessment of

patients undergoing AVR with respect to the possibility of the occurrence of a PA. The degree of calcifications of the aortic root may play an additional role. However, we suggest that the underlying pathology that necessitated aortic valve replacement or composite grafting may well be another important key parameter. Although our data suggest a close association between the etiology that led to the implantation of a composite graft and the development of PA after graft implantation, this relationship does not necessarily imply causality. Location of most PAs at the level of the aortic root underlines the importance of our finding of an increased risk of PA after composite grafting in association with an increased diameter of the aortic root.

Limitations

We are aware of the fact that the patient number of our study was too small to draw any definite conclusions. However, PA is indeed a rather rare complication, and all cases of PA without endocarditis after AVR in our tertiary care institution over a 15-year-period had been taken into account.

We found a remarkable probability to develop PA in patients with composite grafts. Given a relatively large CI, however, the chance for a PA might only be as twice as high as in patients with AVR only.

The significant Fisher's Exact test strongly suggests a close association between the implantation of a composite graft and the location of PA. Using logistic regression, one could exactly quantify this association. However, such an analysis would be worthless in our setting as PA only occurred after the surgical procedure.

The controls for our case-control pairs had been chosen according to age, gender and time of operation using a computer program designed for this purpose. Therefore, it cannot be completely ruled out that the number of patients with composite grafts within our controls might have been low by chance only.

At last, it was beyond the scope of this study to analyze other factors such as a sub-clinical endocarditis, or the surgeon himself, i.e. his experience, and, at least theoretically depending on this, the pump time or any trauma involved. All operations done at our institution were performed by very experienced heart surgeons. However, we feel unable to statistically analyze these factors in a valid way in this retrospective study.

Conclusions

Noninfectious PAs are a rather rare complication in patients after AVR with an incidence in our cohort of <1%. However, in patients with composite grafts and homografts, this value increases to 4%. Unlike the management of infective PAs in a postoperative setting, our data suggest that noninfectious PA is a relatively benign condition, making watchful waiting and repeated echocardiography the procedure of choice in these patients. This recommendation is based on our series, where no patient had to undergo reoperation for PA during the follow-up period of several years. However, PAs were detected after a median of 68 days after AVR. This means that in general PAs develop relatively early in the postoperative period. Based on our data, we therefore suggest the first non-invasive follow-up using TTE three months after AVR, in particular in those patients in whom a composite graft had been inserted.

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Figure legends

Figure 1: Echocardiographic visualization of a pseudoaneurysm in end systole without (A) and with (B) color-mode.

Figure 2: Box plots showing the distribution of diameters of the aortic root (A) and of the ascending aorta (B) in patients with PA previously undergoing composite graft implantation or aortic valve replacement only.

Table 1: Clinical and echocardiographic characteristics of all patients who developed a pseudoaneurysm without a history of endocarditis

Pa t	Gen de r	Age	Cusps	□ AV- Annulu s	□ Aortic root	□ Aorta ascendens	Type of Prosthesis	□ AV- Prosthesis	Localization	Etiology	Time to PA (days)
1	M	60	2	24	39	38	CM	23	NC	bicusp.	54
2	M	60	3	24	N/A	N/A	SJM	23	NC	tricusp.	1
3	M	41	3	24	59	61	CM (Comp.)	23	LC	A-Dissection	63
4	M	57	3	27	74	30	CM (Comp.)	25	AA	A-Dissection	9
5	F	57	0	21	31	40	Shelhigh (Comp.)	21	RC	Aortic insufficiency	73
6	M	39	0	31	50	37	Homograft	28	LC	Aortic insufficiency	216
7	M	20	0	23	26	40	SJM (Comp.)	25	NC/LC	unknown	6289
8	M	47	3	30	59	29	Shelhigh (Comp.)	27	RC	A-Dissection	21
9	F	66	0	29	31	29	CE (Comp.)	29	NC	Sinusrup tur	2611
10	F	67	0	25	51	47	CM (Comp.)	25	AA	Aneurysm ascend.	163
11	M	48	0	29	N/A	N/A	SJM (Comp.)	31	NC	Aortic insufficiency	1
12	F	65	3	21	35	38	CE	21	NC	tricusp.	57
13	M	21	3	23	35	N/A	Homograft	20	NC/RC	Aortic insufficiency	182
14	M	82	3	21	39	37	ELS	23	NC	tricusp.	8
15	M	35	2	39	99	N/A	CM (Comp.)	31	RC	A-Dissection	104
16	M	62	0	21	31	35	Homograft	19	NC	Aortic insufficiency	22
17	F	46	3	24	59	42	SJM (Comp.)	25	RC/LC	Aortic insufficiency	6
18	M	64	3	25	69	25	Composite	25	RC	A-Dissection	56
19	M	87	3	22	31	28	CE	21	RC/NC	tricusp.	192
20	M	50	0	22	36	44	Shelhigh (Comp.)	21	NC	Aortic insufficiency	15
21	M	30	2	27	38	42	CM	25	LC	bicusp.	1310
22	F	58	3	26	63	43	Shelhigh (Comp.)	25	NC	A-Dissection	581
23	M	47	0	29	31	31	CM	25	RC	Aortic insufficiency	3425
24	M	62	3	24	67	N/A	SJM (Comp.)	29	RC	A-Dissection	1440

Pat = Patient.

Cusps = Number of Cusps of AV, 0 indicates replaced AV during previous AV-operation.

□ = Diameter in Millimetre.

Type of prosthesis: CE = Carpentier Edwards. CM = Carbomedics. ELS = Edwards Life Science. SJM = St. Jude Medical. (Comp.) = Composite graft.

N/A = not measured at time of echocardiography.

Localization: NC = non-coronary aortic sinus. LC = left coronary aortic sinus. RC = right coronary aortic sinus.

Etiology: Bicusp. = combined stenotic and insufficient bicuspid aortic valve. Tricusp. = combined stenotic and insufficient tricuspid aortic valve. A-Dissection = aortic dissection (Stanford classification A). Sinusrup tur = Rupture of the sinus of valsalva. Aortic insufficiency = Aortic valve insufficiency. Aneurysm ascend. = Aneurysm of the ascending aorta

Figure 1:

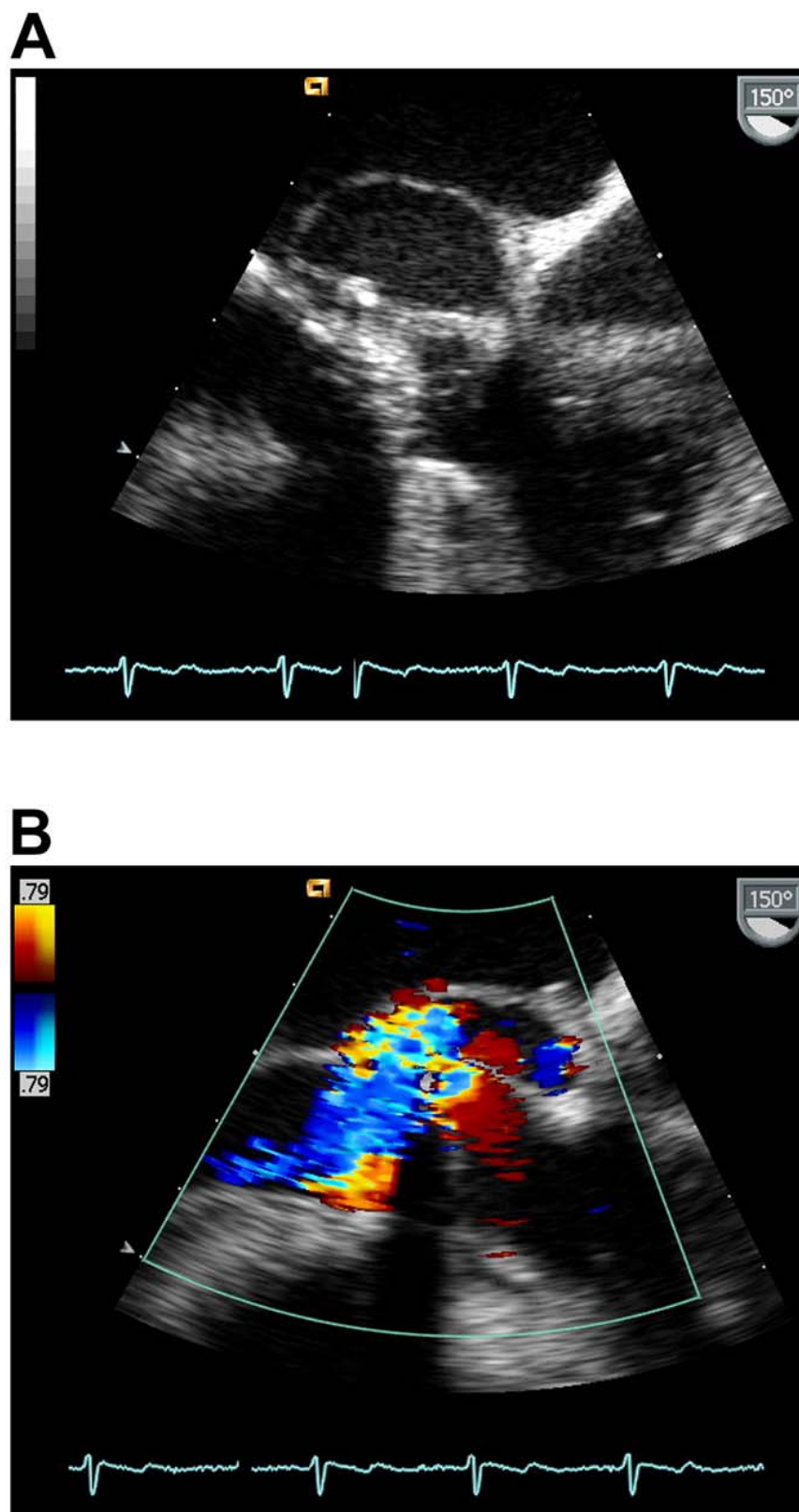
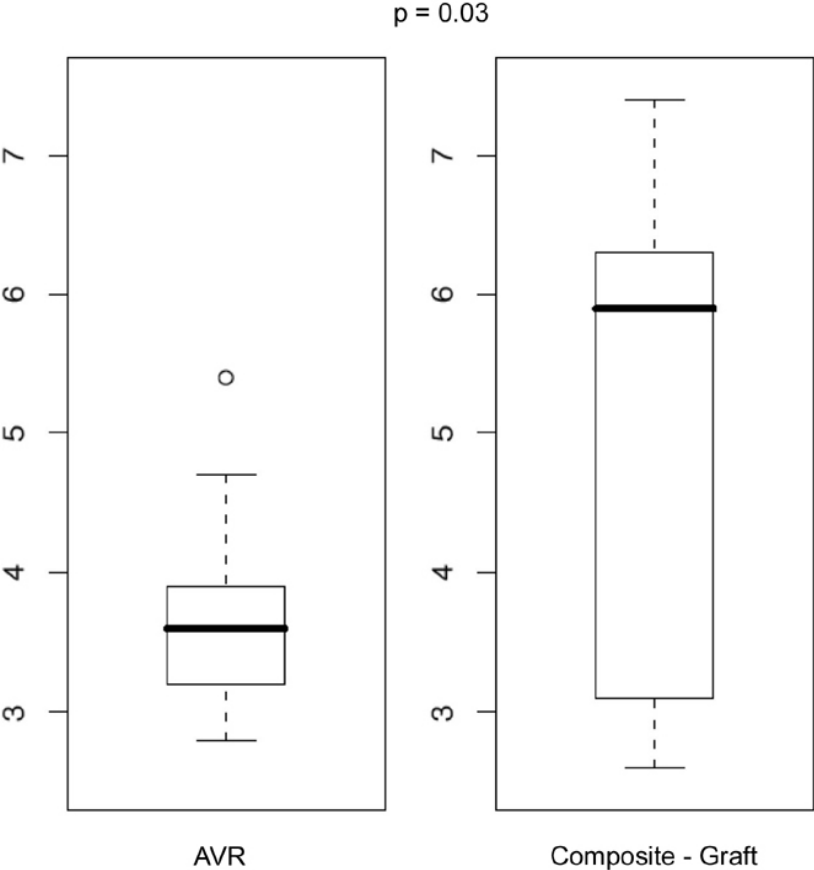


Figure 2:

A aortic root



B ascending aorta

